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## **Critical Care**

**TOPIC:** Critical Care

TYPE: Medical Student/Resident Case Reports

## HYPOCORTISOLISM IN A PATIENT WITH COVID-19: A CASE REPORT AND DISCUSSION ON MANAGEMENT

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**INTRODUCTION:** Long COVID- 19 or post-acute COVID -19 syndrome is now recognized as a range of symptoms that can last weeks or months after first being infected with COVID-19 or SARS -CoV 2 virus. It can appear weeks after infection irrespective of the severity of the illness1. It can affect most, if not all, body systems including heart, lung, kidney, skin, and brain functions. Here we present a case with Covid-19 infection with hyporcortisolism presenting as dyspnea and fatigue as the dominant long Covid-19 symptom.

CASE PRESENTATION: A 55-year-old female with a past medical history of hyperthyroidism on propranolol, was diagnosed with mild COVID -19 infection, advised to treat with self-isolation. She revisited ER 6 weeks later with persistent dyspnea, fatigue, and poor ability to focus and concentrate. She was given a trial of the direct oral anticoagulant for 4 weeks with no change in her symptoms. The subsequent workup done; head-computer tomography scan (CTS) was negative for subarachnoid hemorrhage and cerebral venous thrombosis. The Lungs CTS did not reveal any pulmonary fibrosis. The 6-minute walk test was impaired at 79%. The Pulmonary Function test did not show any significant obstruction or restriction. The Cardiopulmonary Exercise Test overall was suggestive of deconditioning. The laboratory work included creatine kinase, Aldolase, vitamin B12, Folic acid, C-reactive protein, Vitamin D, Antinuclear antibody Thyroid Function test all were unremarkable. The AM cortisol level came back as less than 2. The patient was started on oral prednisone for a diagnosis of relative adrenal insufficiency. All her symptoms improved within 2 weeks of treatment and resolved after 4 -6 weeks, she was tapered down and finally weaned off prednisone.

**DISCUSSION:** The proposed mechanism of COVID-19 induced relative adrenal insufficiency is likely hypophysitis.2 The principal target for the virus entry is the angiotensin-converting Enzyme 2 (ACE-2) receptor which is found on pneumocytes, arterial and venous endothelial cells of many organs including adrenal glands and hypothalamic and pituitary tissues2. Furthermore, SARS- Cov2 virus has amino acid sequences which are similar to the host's ACTH, so antibodies made against the virus destroys the host ACTH and consequently may cause contribute to cortisol insufficiency.2,3

**CONCLUSIONS:** Post COVID-19 infection has a multifaceted manifestation. Hypocortisolism can persist in patients with post COVID-19 and should be sought for if the symptoms are suggestive. The treatment is simple and effective.

**REFERENCE #1:** Naserghandi, S.F. Allameh and R. Saffarpour. All about COVID-19 in brief. New Microbes New Infect. 2020 May; 35: 100678. Published online 2020 Apr 13. doi: 10.1016/j.nmni.2020.100678 PMID: 32292590.

**REFERENCE #2:** Pal, R. COVID-19, hypothalamo-pituitary-adrenal axis and clinical implications. Endocrine 68, 251–252 (2020). https://doi.org/10.1007/s12020-020-02325-1.

**REFERENCE #3:** R. Wheatland, Molecular mimicry of ACTH in SARS—implications for corticosteroid treatment and prophylaxis. Med. Hypoth. 63, 855–862 (2004) PMID: 15488660.

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